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## 2.4 BRACHIAL CUFF RESERVOIR CHARACTERISTICS AND END-ORGAN MARKERS OF CARDIOVASCULAR RISK IN AUSTRALIAN ADULTS: A CROSS-SECTIONAL STUDY

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**Objective:** Reservoir-excess pressure measured using tonometry methods predicts cardiovascular events, but the operator-dependency of tonometry is an impediment to widespread use. A cuff-based blood pressure device has been developed to derive reservoir-excess pressure from measured brachial pressure waveforms, but whether this method is independently associated with cardiovascular risk has never been investigated and this was the aim of this study.

**Methods:** 1874 adult participants (age  $43.7 \pm 5.2$  years, 11% male) from the Longitudinal Study of Australian Children's Child Health CheckPoint study had reservoir pressure (RP) and excess pressure (XSP) derived from the brachial pressure waveform measured using cuff oscillometry (SphygmoCor XCEL, AtCor Medical, Sydney).

Central hemodynamics (augmentation index and central blood pressure) were estimated from the central pressure waveform. Carotid intima-media thickness (cIMT,  $n = 1467$ ) and carotid-to-femoral pulse wave velocity (cf-PWV,  $n = 1674$ ) were measured as end-organ markers of cardiovascular risk. **Results:** XSP and RP were associated with cIMT after adjusting for age, sex, waist-to-hip ratio, heart rate (HR) and central hemodynamic indices ( $\beta = 0.070$ ,  $p = 0.027$  and  $\beta = 0.052$ ,  $p = 0.047$ ). RP was also significantly associated with cf-PWV after adjusting for the same variables as above ( $\beta = 0.128$ ,  $p < 0.001$ ). The additional reservoir-excess pressure variables in a model that originally included the Framingham risk score and HR strengthened the evidence for associations with cIMT and cf-PWV ( $p < 0.001$  for all  $R^2$  changes).

**Conclusion:** Cuff-based measures of reservoir-excess pressure are significantly associated with end-organ markers of cardiovascular risk independent of traditional risk factors. This cuff method may provide additional information to improve cardiovascular risk stratification.

## 2.5 NON-INVASIVE WAVE INTENSITY ANALYSIS IN THE AORTA AND INTERNAL CAROTID USING PHASE-CONTRAST MR ANGIOGRAPHY: THE EFFECT OF HYPERTENSION

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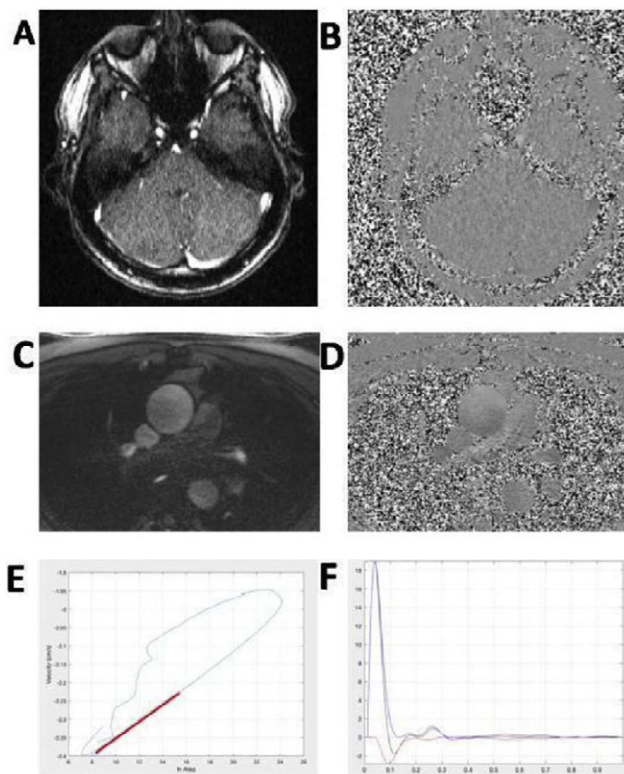
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**Introduction:** Hypertension is associated with stiffening of blood vessels, reduced arterial lumen and reduced cerebral blood flow; however, it is not known how lower cerebral blood flow relates to arterial structure or impacts on wave dynamics. We hypothesise increased backward wave energy and faster wave speed in the hypertensive internal carotid artery as an indication of increased resistance to flow.

**Methods:** Normotensive, controlled and uncontrolled hypertensive participants were recruited (daytime ambulatory BP  $< 135/85$  mmHg and  $> 135/85$  mmHg, respectively;  $n = 11$  per group). Wave intensity analysis was performed on left internal carotid and ascending aorta phase-contrast magnetic resonance angiography.

**Results:** While ascending aortic wave speed increased significantly in the uncontrolled hypertensive compared to normotensive ( $p < 0.001$ ) and controlled hypertensive participants ( $p = 0.038$ ), no significant difference was observed in the internal carotid. Carotid forward and backward wave intensity increased in uncontrolled hypertensives compared to normotensives ( $p = 0.036$  and  $p = 0.033$ , respectively), and backward wave energy increased in the controlled hypertensives compared to normotensives ( $p = 0.041$ ). There was no significant difference between uncontrolled and controlled hypertensives.



**Figure 1** Analysis of the phase contrast MR angiography data. A) Magnitude image B) Phase image of the internal carotid arteries C) Magnitude image and D) Phase image of the ascending aorta E) example of  $\log(\text{Area})$ -Velocity loop. Red line indicates the slope from which wave speed is calculated in early systole F) Example of the wave intensity components, where blue is the forward wave energy, red is the backward wave energy and black is the net wave intensity.

**Conclusion:** Wave intensity in the internal carotid artery is altered in uncontrolled hypertension. This is partly rescued when blood pressure is controlled by medication, although greater backward wave energy persists. This supports the hypothesis of increased resistance to flow in the cerebral circulation of the hypertensives. Whilst increased aortic wave speed confirmed an expected increase in stiffness, this was not observed in the internal carotid. This might suggest a protective mechanism in the cerebral circulation, in conjunction with the effect of vessel tortuosity.

## 2.6 BLOOD PRESSURE-INDEPENDENCE OF AORTIC-TO-BRACHIAL ARTERY STIFFNESS RATIO IS DEPENDENT ON DISEASE STATUS

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**Introduction:** Aortic stiffness predicts cardiovascular mortality but is limited as a risk marker because it is dependent on blood pressure (BP). A potential solution is provided from the ratio of aortic-to-brachial artery stiffness (ab-ratio), which is purported to be a BP-independent risk marker among patients with renal dysfunction (RD). We sought to determine the BP-independence of the ab-ratio in patients with disease (including RD) and healthy populations.

**Methods:** The ab-ratio (aortic/brachial pulse wave velocity; PWV) and mean arterial pressure (MAP) were recorded in patients with RD ( $n = 119$ , aged  $65 \pm 7$  years), hypertension ( $n = 140$ , aged  $62 \pm 9$  years), type 2 diabetes ( $n = 77$ , aged  $60 \pm 9$  years) and healthy individuals ( $n = 99$ , aged  $51 \pm 8$  years). Multiple-regression analysis was performed to test the independent association of MAP with the ab-ratio adjusted for age, sex, body-mass index and blood glucose.

**Results:** There was no significant relationship between the ab-ratio and MAP in patients with RD ( $\beta = 0.002$ , 95% CI 0.002, 0.006,  $p = 0.34$ ), hypertension ( $\beta = 0.001$ , 95% CI 0.003, 0.006,  $p = 0.62$ ) or diabetes ( $\beta = 0.006$ , 95% CI 0.002, 0.014,  $p = 0.11$ ). However, in healthy individuals the ab-ratio was